

Significance of asymmetrically inverted T wave

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SUMMARY Two consecutive series of patients with a T wave asymmetry ratio of 2.0 or greater have been studied. Patients with bundle-branch block or who were on digoxin or a similar drug were excluded. In 50 of the 69 patients, the heart was examined either by echocardiography or by direct inspection.

Sixty-one of the 69 patients had diseases commonly associated with left (or right) ventricular hypertrophy and/or dilatation. The remaining eight patients had clinically pure ischaemic heart disease.

Of the 50 hearts examined by echocardiography or direct inspection (including six with pure ischaemic heart disease), 49 were found to have abnormal thickness of the left (or right) ventricle, or increased end-diastolic left ventricular diameter, or a combination of hypertrophy and dilatation.

In 12 of the 47 patients with left ventricular hypertrophy or dilatation, the electrocardiogram did not satisfy the Sokolow and Lyon voltage criterion of left ventricular hypertrophy.

T wave inversion in the lateral leads of the electrocardiogram presents a continuing problem in interpretation.¹ It is frequently impossible to be sure whether it indicates ischaemia or left ventricular hypertrophy. There has long been an impression that the inverted T wave in left ventricular hypertrophy is less symmetrical than that in ischaemia²; but this has never been established. Unfortunately, the early descriptions of the repolarisation pattern in left ventricular hypertrophy were confused and conflicting, and there does not seem to have been any attempt to quantify the degree of T wave asymmetry. Furthermore, there was at that time no means of measuring the dimensions of the left ventricular wall and cavity size during life nor of excluding the presence of coexisting coronary narrowing. As a result, the shape of the inverted T wave never became established as evidence of ventricular hypertrophy. Indeed, most modern textbooks on clinical electrocardiography state or imply that the repolarisation patterns in coronary disease and left ventricular hypertrophy are indistinguishable.

Now, thanks to echocardiography, we have a means of assessing the degree of left ventricular thickness and dilatation during life. We also have a method of measuring the degree of T wave asymmetry³ as the ratio between the angle of descent (a) and the angle of ascent (b) (Fig.). We have therefore undertaken a study in which consecutive patients with a specified

degree of asymmetry of the inverted T wave were subjected to a careful examination of the cardiovascular system including echocardiographic or direct visual assessment of the state of the heart.

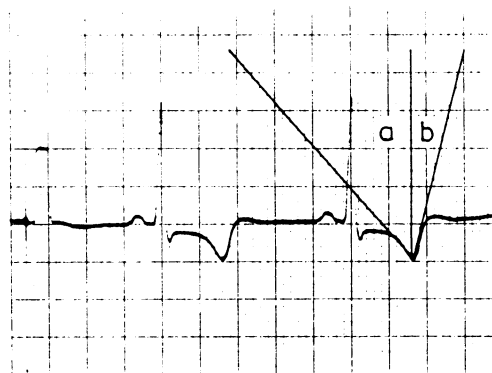


Fig. Asymmetrically inverted T waves. Lead I from a man aged 50 with total occlusion of the right and narrowing of two branches of the left coronary artery. He had been normotensive for at least the previous 13 years and had no evidence of valvular disease. The asymmetry ratio (a/b) is 2.7. Echocardiography showed the thickness of the free wall of the left ventricle to be 13 mm and that of the septum 21 mm (upper limit of normal of both measurements, 12 mm). The end-diastolic diameter was 44 mm (within normal limits).

Subjects and methods

SERIES A

In a previous study,⁴ 100 consecutive patients referred to a cardiac unit or seen in private cardiological practice with T inversion of 1 mm or more in V6 were analysed, and the repolarisation pattern in this lead was correlated with the diagnosis. (Patients with bundle-branch block and those receiving digoxin or any similar drugs were excluded.) From this series, we extracted all those with a T wave asymmetry ratio of 2.0 or greater. Twenty-eight patients fulfilled this criterion.

SERIES B

After the conclusion of Series A, we broadened our criteria of acceptance to include patients with an asymmetry ratio of 2.0 or greater in any lead. As before, the only patients excluded were those with bundle-branch block and those on digoxin or a similar drug. This series contains a further 41 patients.

SERIES A AND B

Of the 69 patients, 44 were male and 25 female. Their ages ranged from 17 to 91 years with a mean of 60 years.

Each patient had at least two 12 lead electrocardiograms and one departmental chest x-ray recorded. Twenty-four patients were studied by coronary arteriography and left heart catheterisation and two by right heart catheterisation. In addition, the hearts of two other patients were inspected directly, one at operation and the other at necropsy.

The asymmetry ratio was calculated as previously described.³ In Series B, the ratio was calculated in the lead which showed the greatest degree of asymmetry. This was V6 in 14 cases, lead I in nine, lead II in six, lead V5 in five, lead V4 in three, lead VL in two, lead V3 in one, and lead V2 in one. (In patients with several electrocardiograms, the degree of asymmetry varied to some extent from month to month and even from day to day, as previously noted.⁴ The RS voltage in the precordial leads was measured by the method of Sokolow and Lyon.⁵ A diagnosis of ischaemic heart disease was based on the presence of either unequivocal evidence (historical, electrocardiographic, enzymatic, or necropsy) of myocardial infarction, or the demonstration of 70% narrowing in at least one main coronary artery.

Forty-eight patients were studied by echocardiography. All the echocardiograms were performed by the same operator (JW), from the standard left parasternal approach. The majority of patients had recordings taken from a Picker A Scan attached to a Cambridge multichannel recorder with a paper speed of 50 mm per second. The last 12 patients had echocardi-

grams performed using an ATL Series 300 C, again with a paper speed of 50 mm per second. All the echocardiograms were technically satisfactory in that the interventricular septum and posterior left ventricular wall were clearly seen just below the level of the free cusp edge of the mitral valve.

The wall thicknesses and the left ventricular intracavity diastolic dimension were measured at end-diastole, being standardised to occur at the upstroke of the R wave of the electrocardiogram. The upper limit of normal for the interventricular septum and the posterior left ventricular wall was 12 mm and for the intracavity dimension at end-diastole it was 56 mm. Left ventricular mass was calculated using the Penn convention measurements in the equation $LVM = 1.04 [(LVIDp + PWTp + IVSp)^3 - (LVIDp)^3] - 13.6 \text{ g}$.^{6,7} An upper limit of normal of 200 g was accepted.

Results

CLINICAL DIAGNOSIS IN PATIENTS WITH T WAVE ASYMMETRY RATIO OF 2.0 OR MORE (Table 1)

Eighteen patients had apparently pure aortic valve disease, and three a combination of aortic valve disease and ischaemic heart disease. Sixteen had apparently pure hypertension, and nine a combination of hypertension and ischaemic heart disease. Eight had some combination of aortic valve disease, hypertension and mitral regurgitation. Five had hypertrophic cardiomyopathy and eight had apparently pure ischaemic heart disease. One patient had severe pulmonary valvar stenosis and another primary pulmonary hypertension.

FINDINGS AT ECHOCARDIOGRAPHY OR ON DIRECT INSPECTION IN PATIENTS WITH T WAVE ASYMMETRY RATIO OF 2.0 OR MORE (Table 2)

Forty-eight patients were studied by echocardiogra-

Table 1 *Diagnostic analysis of 69 patients with an asymmetry ratio of 2.0 or more*

Diagnosis	Series		
	A	B	A+B
Aortic valve disease, pure	10	8	18
Aortic valve disease associated with ischaemic heart disease	3	0	3
Hypertension, pure	7	9	16
Hypertension associated with ischaemic heart disease	1	8	9
Mitral regurgitation, aortic valve disease, hypertension	3	5	8
Hypertrophic obstructive cardiomyopathy	2	3	5
Pure ischaemic heart disease	2	6	8
Other	0	2	2
Totals	28	41	69

Table 2 *Echocardiographic findings in 48 patients with an asymmetry ratio of 2.0 or more*

Diagnosis	No.	Wall over 12 mm	Diameter over 55 mm	Wall and diameter increased	LV mass ≥200 g	No abnormality
Aortic valve disease, pure	13	6(3)	2	5	13(3)	0
Aortic valve disease and ischaemic heart disease	2	1	1	0	2	0
Hypertension, pure	9	7(1)	0	2(1)	9(2)	0
Hypertension and ischaemic heart disease	7	1(1)	2(1)	3(1)	6(2)	1(1)
Mitral regurgitation, aortic valve disease, hypertension	6	3	2	1	6	0
Hypertrophic obstructive cardiomyopathy	4	4(1)	0	0	3	0
Pure ischaemic heart disease	6	3(1)	2	1(1)	6(2)	0
Other	1	RV+	0	0	0	0
Totals	48	26(7)	9(1)	12(3)	45(9)	1(1)

Figures in brackets indicate cases without Sokolow and Lyon criterion of left ventricular hypertrophy.

phy. One of these had pure right ventricular hypertrophy associated with pulmonary hypertension. The remaining 47 patients had T wave inversion in one or more left ventricular leads. In 46 of these, the left ventricular wall or septum was over 12 mm thick or the end-diastolic diameter was over 56 mm. The one patient who showed no definite left ventricular abnormality was a woman whose wall thickness was at the upper limit of normal (12 mm). Forty-five of the 47 patients had a calculated left ventricular mass above the accepted upper limit of normal (200 g).

In two patients, the heart was inspected directly. One patient was examined at necropsy and found to have left ventricular hypertrophy with a heart weighing 500 g. The other, a patient with pulmonary stenosis, was noted to have right ventricular hypertrophy at the time of operation.

Thus, all but one of the 50 patients studied by echocardiography or direct inspection were found to have an abnormally thick ventricle or an abnormally large end-diastolic ventricular diameter.

The Sokolow and Lyon⁵ criterion of left ventricular hypertrophy was fulfilled in 27 of the 38 patients with echocardiographic or necropsy evidence of left ventricular hypertrophy and in eight of the nine patients with only an abnormally large end-diastolic diameter.

Discussion

The association of an asymmetrically inverted T wave with left ventricular hypertrophy was recorded many years ago² but has latterly been ignored. It is only relatively recently that it has been possible to assess the specificity of this pattern during life by determining the thickness of the left ventricular wall with reasonable accuracy by means of echocardiography and by excluding coronary narrowing as a contributory factor by means of coronary arteriography. In a previous paper³ it was shown that the asymmetrical T wave was found in patients with aortic valve disease without significant coronary disease. In the present

study we have correlated this electrocardiographic pattern with the state of the left ventricle as determined by echocardiography.

Echocardiography has been shown to provide a reliable measure of the thickness of the left ventricular wall when compared with both surgery and necropsy⁸ and with angiography.⁹ The upper limit of 12 mm for both septal and posterior wall thickness in adults has been accepted for a number of years.¹⁰ The end-diastolic diameter as measured at echocardiography represents the minor axis of the left ventricle and has been shown to correlate closely with other measurement techniques.¹¹⁻¹³ Fifty-six mm is the accepted upper limit of normal end-diastolic left ventricular dimension in adults.¹⁰ Admittedly, some workers¹⁴ have reported considerable change in left ventricular end-diastolic diameter (up to 10.5 mm) without change in heart rate; but most accept a variability of only 3 or 4 mm.^{8 10 15}

This study indicates a strong association between pronounced asymmetry of the inverted T wave in any lead and the presence of ventricular hypertrophy or dilatation. Of the 69 patients with pronounced asymmetry of the inverted T wave, 61 had diseases that are characterised by ventricular hypertrophy. The remaining eight had ischaemic heart disease; but the seven of these who were studied by either echocardiography or at necropsy were all found to have left ventricular hypertrophy or an abnormally large end-diastolic diameter. Altogether, 49 of the 50 patients studied by echocardiography or direct inspection were shown to have an abnormal ventricular thickness or increased end-diastolic diameter. The single exception was a woman with hypertensive and ischaemic heart disease whose left ventricular thickness was at the upper limit of normal (namely 12 mm). Of the 47 patients with T wave inversion in one or more of the left ventricular leads, studied by echocardiography, 45 had a calculated left ventricular mass above the accepted upper limit of normal (200 g).

Twelve of the 47 patients shown to have abnormal

left ventricular wall thickness or increased end-diastolic diameter did not fulfil the Sokolow and Lyon⁵ voltage criterion of left ventricular hypertrophy. Admittedly this criterion is imperfect. It is, nevertheless, the generally accepted standard¹⁶ and there is no agreement with regard to a better one.

A T wave asymmetry ratio of 2.0 or more may be regarded as an additional independent indicator of ventricular hypertrophy or dilatation.

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